phila has not been identified yet. Indeed, the formin we identified as Drok substrate genetically interacts with Drok and its knock-down causes PCP phenotypes.

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Program/Abstract # 205
The roles of beta-catenin pathway in the chick dermomyotome and myotome
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Cell–cell signaling through extracellular signals is essential for development. Wnts are a family of secreted proteins important for their roles in proliferation and differentiation in both vertebrates and invertebrates. We have previously shown that β-catenin dependent Wnt signaling (via Wnt3a) causes an expansion of the dermomyotome and myotome. In these studies, we showed that tissue proliferation in the dermomyotome coupled with cell hypertrophy in the myotome contributed to the expansion of the myotome. However, we did not assay the requirement of β-catenin dependent Wnt signaling in myogenesis. We hypothesized that β-catenin signaling is required for proper development of the dermomyotome and myotome. To test our hypothesis, we overexpressed Dickkopf1 (Dkk1), a known inhibitor of the Wnt/β-catenin pathway in the neural tube of developing chick embryos and assayed the effects on the dermomyotome and myotome. Our results showed that over expression of Dkk1 reduced the area of the dermomyotome and myotome, as compared to controls. Many mechanisms could explain the reduction in size of the myotome. To determine if myogenesis was delayed by Dkk1, we performed whole mount immunostaining of electroporated embryos with antibodies against Myosin Heavy Chain (MHC). Our preliminary results indicated that MHC expression on the electroporated side is delayed by ~1.1 somites compared to the control side ($n=8$, p-value <0.05). We are currently working to distinguish whether this is an effect on specification and/or differentiation.

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Program/Abstract # 206
Role of Ror1 in the developing chick neural tube and somites
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Many congenital diseases occur due to defects in neural tube (NT) and somite development. One family of secreted proteins that plays a major role in these processes are Wnts. Though Wnts usually signal via Frizzled receptors, Ror1 and Ror2 tyrosine kinase receptors have also been shown to play important roles in Wnt signaling. They have been shown to activate both the β-catenin dependent and independent pathways. For example, Wnt5a signaling via Ror1/2 inhibits the β-catenin dependent pathway while Wnt3a signaling via Ror2 activates the β-catenin dependent pathway. This implies that depending on which Wnt is bound to the receptor, different pathways can be excited. Our data shows Ror1 expression in the dorsal NT and throughout the dermomyotome of developing chick embryos. Together, these data led us to hypothesize that Ror1 has a key role in the NT and somites. Our research goal is to determine the role of Ror1 and to distinguish whether it acts via a β-catenin dependent or independent pathway. To test the role of Ror1, we knocked it down in the NT and somites by electroporating an RNAi construct. Because the area of the NT and somites was noticeably reduced on the electroporated side as compared to controls, we assayed for apoptosis using a TUNEL assay. Cell death was seen on the electroporated side of the NT but not in controls. By contrast, no increase in cell death was observed in electroporated somites. We are currently testing whether Ror1 acts via a β-catenin dependent or independent pathway in the NT.

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Program/Abstract # 207
Restricting cell movement: The role of Tspan18 in neural crest migration
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Unlike typical neuroepithelial cells in the developing central nervous system, neural crest cells undergo an epithelial to mesenchymal transition (EMT) and migrate great distances to give rise to diverse structures, such as the peripheral nervous system, melanocytes and facial bone. Anomalies of the neural crest lead to several cancers including neuroblastoma and melanoma, and metastatic cells undergo an EMT that resembles predictable events during neural crest delamination, making the neural crest a unique model to study cancer progression. Despite its fundamental importance, neural crest delamination and migration are poorly understood. Tetraspanin 18 (Tspan18), a member of the tetraspanin family of transmembrane proteins whose activity has been implicated in cell signaling, motility and adhesion, is abundantly and specifically expressed in premigratory chick neural crest cells. Interestingly, Tspan18 expression is down-regulated when neural crest cells migrate, suggesting that Tspan18 negatively regulates neural crest migration. Interfering with Tspan18 function promotes, while overexpression inhibits neural crest cell migration. These data suggest that Tspan18 may play a vital role in neural crest emigration from the neural tube, regulating the proper developmental timing of neural crest migration. Gaining insight into Tspan18 function in neural crest cells will give us a better understanding of how dynamic interactions at the cell surface mediate cell migration, and may provide clues to understanding how cancers like neuroblastoma become metastatic.

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Program/Abstract # 208
The Twist-Slug-Snail regulated gene sizzled's role in mesoderm and neural crest formation
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The secreted Wnt and Tolloid proteinase inhibitor Sizzled (Salic et al., 1997. Development **124**:4739; Lee et al., 2006. Cell **124**:147) is an immediate-early target of regulation of the transcription factors Slug/Snail2 and Twist in the early *Xenopus* embryo. Similarly, the level of *sizzled* RNA increases in Snail1 morphant embryos. An in situ hybridization analysis indicates that in addition to its previously described expression in the ventral regions of blastula stage embryos, *sizzled* expression is present at the anterior (cement gland) and posterior (proctodeum) domains of neurula stage embryos, and can